IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

IN RE APPLICANT	: Jackowski *et al.*
INVENTION	: **Protein Biopolymer Markers Indicative of Alzheimer's Disease**
SERIAL NUMBER	: 09/992,672
FILING DATE	: November 23, 2001
EXAMINER	: Cook, Lisa V.
GROUP ART UNIT	: 1641
OUR FILE NO.	: 2132.088

CERTIFICATE UNDER 37 CFR 1.8(a)
I hereby certify that this correspondence is being deposited with the U.S. Postal Service as First Class mail in an envelope addressed to Commissioner for Patents P.O. Box 1450, Alexandria, VA 22313-1450 on 11-7-2005

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Alexandria, VA 22313-1450

DECLARATION UNDER 37 CFR § 1.132

I, Ferris H. Lander, do hereby declare as follows:

1. I am a registered Patent Agent and am authorized to represent the inventor's and assignee in the application entitled **"Protein Biopolymer Markers Indicative of Alzheimer's Disease"**, having U.S. Application Serial No. 09/992,672, filed November 23, 2001.

2. In the Office Action mailed on August 9, 2005, claim 1 (as presented on March 31, 2005) was rejected under 35 USC 101 because the claimed invention allegedly is not supported by either a

specific, substantial, credible or asserted utility or a well-established utility. Claim 1 was also rejected under 35 U.S.C. 112, first paragraph because the claimed invention allegedly contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

Specifically, the Examiner asserts that the figures do not identify SEQ ID NO:3 (the claimed biopolymer marker) so it (SEQ ID NO:3) is not proven to be differentially expressed in any of the samples.

3. The attached figure was produced by scanning the original photograph of the gel. The figure is entitled "DEAE 3(Elution) AD vs. Age Matched AD (Control)" and represents Figure 3 as originally filed. No new matter has been added; this figure is simply a clearer copy of Figure 3 as originally filed and is provided to clarify the presence and differential expression of SEQ ID NO:3 (the claimed biopolymer marker). The gel shown in the attached figure does not represent new experimentation; the figure shows a clearer image of the original gel made at the time that the experiments described in the instant specification were first carried out.

The undersigned declares that all statements made herein of his own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the Application or any patent issuing thereon.

11/7/2005
Date

Ferris H. Lander
Reg. No. 43,377

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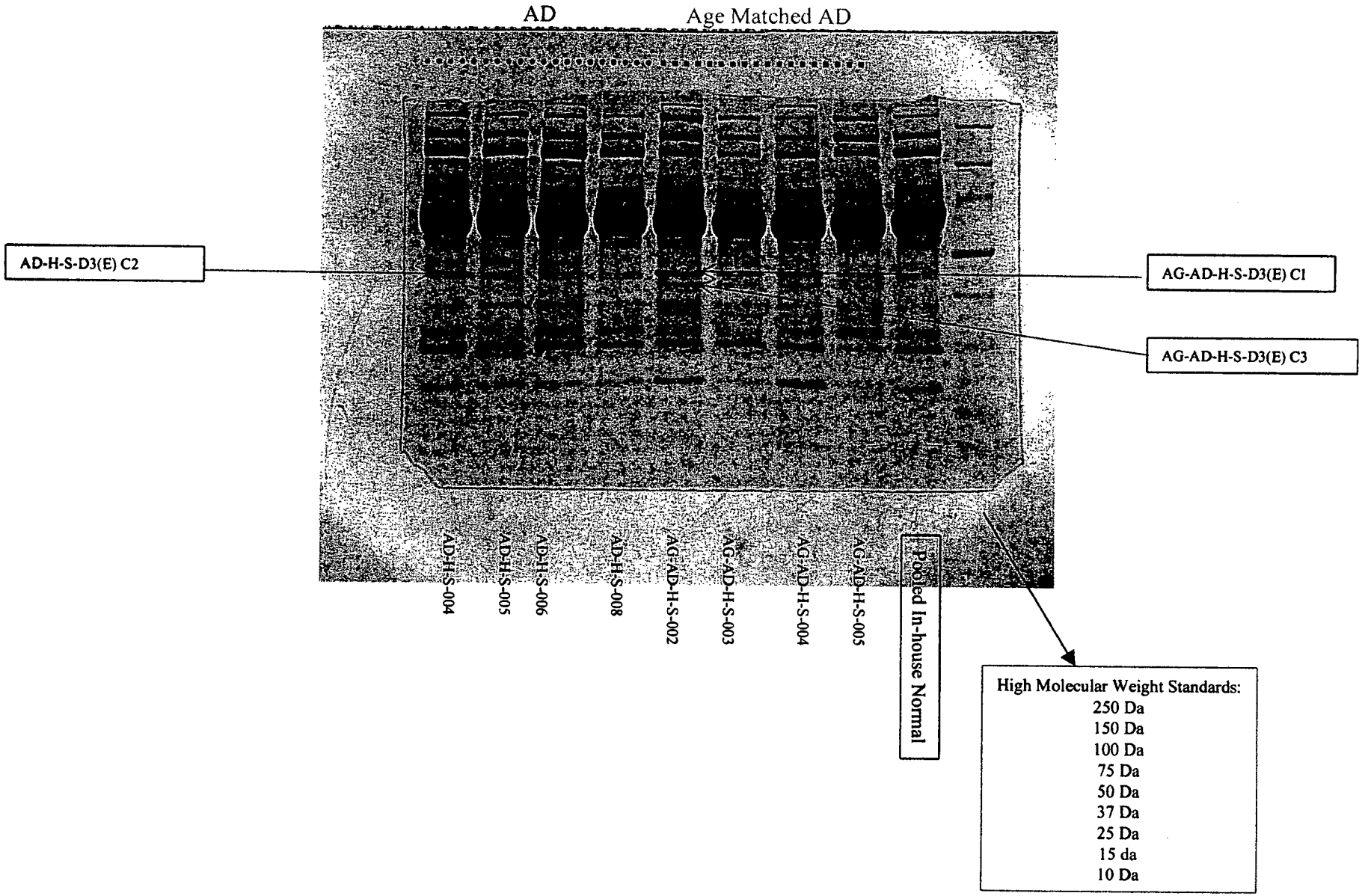
DEAE 3(Elution) AD vs. Age Matched AD (Control)



2132.088 Examiner's copy reference # 1

1: Arch Neurol. 1999 Jun;56(6):673-80.

Comment in:
- Arch Neurol. 1999 Jun;56(6):655-6.

Cerebrospinal fluid beta-amyloid(1-42) in Alzheimer disease: differences between early- and late-onset Alzheimer disease and stability during the course of disease.

Andreasen N, Hesse C, Davidsson P, Minthon L, Wallin A, Winblad B, Vanderstichele H, Vanmechelen E, Blennow K.

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OBJECTIVES: To study the diagnostic potential of the 42 amino acid form of beta-amyloid (beta-amyloid(1-42)) in cerebrospinal fluid (CSF) as a biochemical marker for Alzheimer disease (AD), the intra-individual biological variation of CSF-beta-amyloid(1-42) level in patients with AD, and the possible effects of differential binding between beta-amyloid and apolipoprotein E isoforms on CSF-beta-amyloid(1-42) levels. DESIGN: A 20-month prospective follow-up study. SETTING: Community population-based sample of consecutive patients with AD referred to the Pitea River Valley Hospital, Pitea, Sweden. PATIENTS: Fifty-three patients with AD (mean +/- SD age, 71.4 +/- 7.4 years) diagnosed according to the National Institute of Neurological and Communicative Disorders and Stroke and Alzheimer's Disease and Related Disorders Association criteria and 21 healthy, age-matched (mean +/- SD age, 68.8 +/- 8.0 years) control subjects. MAIN OUTCOME MEASURES: Cerebrospinal fluid beta-amyloid(1-42) level--analyzed using enzyme-linked immunosorbent assay--and severity of dementia--analyzed using the Mini-Mental State Examination. RESULTS: Mean +/- SD levels of CSF-beta-amyloid(1-42) were decreased (P<.001) in patients with AD (709 +/- 304 pg/mL) compared with controls (1678 +/- 436 pg/mL). Most patients with AD (49 [92%] of 53 patients) had reduced levels (<1130 pg/mL). A highly significant correlation (r = 0.90; P<.001) between baseline and 1-year follow-up CSF-beta-amyloid(1-42) levels was

found. There were no significant correlations between CSF-beta-amyloid(1-42) level and duration (r = -0.16) or severity (r = -0.02) of dementia. Low levels were also found in patients with mild dementia (Mini-Mental State Examination score, >25). CONCLUSIONS: The sensitivity of CSF-beta-amyloid(1-42) level as a diagnostic marker for AD is high. The intra-individual biological variation in CSF-beta-amyloid(1-42) level is low. Low CSF-beta-amyloid(1-42) levels are also found in the earlier stages of dementia in patients with AD. These findings suggest that CSF-beta-amyloid(1-42) analyses may be of value in the clinical diagnosis of AD, especially in the early course of the disease, when drug therapy may have the greatest potential of being effective but clinical diagnosis is particularly difficult.

PMID: 10369305 [PubMed - indexed for MEDLINE]
